
ELECTRORETINOGRAPHIC FINDINGS IN FUCHS' HETEROCHROMIC CYCLITIS

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SUMMARY

Purpose: Fuchs' heterochromic cyclitis (FHC) is an inflammatory disease of unknown aetiology. Although anterior segment signs and vitreous changes are well recognised, retinal features are unusual. To assess the extent of retinal involvement, we performed electrophysiological testing in a group of FHC patients.

Methods: Retinal function was assessed by means of flash electroretinogram (ERG) using a Ganzfeld stimulus, and pattern electroretinogram (PERG) using a checkerboard stimulus of spatial frequency 0.5 and 1.0 cycle per degree reversing at 6 Hz. A total of 21 patients with unilateral, normotensive FHC with visual acuities of 6/5 to 6/9 were studied.

Results: In the flash ERG, selective scotopic b-wave abnormalities occurred in 9 (43%) of 21 FHC eyes. Despite clear media and no history of ocular surgery, 7 patients showed abnormalities of the PERG.

Conclusions: These electrophysiological findings suggest subclinical damage to the inner retinal layers, but not involving the photoreceptors, in eyes with FHC.

Fuchs' heterochromic cyclitis (FHC) accounts for approximately 3% of the total cases of uveitis.¹ Although various theories have been proposed its aetiopathogenesis remains unknown. The clinical spectrum may be a secondary response to a variety of different aetiological agents,² with the triggering stimulus being possibly immunological,³⁻⁵ infectious,^{2,6-10} or a combination of both. Anterior segment features have been well described, including characteristic stellate keratic precipitates, heterochromia, and iris stromal atrophy. There is an absence of posterior synechiae, and vitreous inflammation frequently occurs.¹¹ Although retinal changes are unusual, chorioretinal scars resembling ocular toxoplasmosis have been reported in 7.5-60% of patients.⁹

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The electroretinogram (ERG) is known to provide useful information on retinal function. It can detect retinal damage secondary to vasculitis, uveitis and retinitis. Changes in the potentials arising in the photoreceptors and pigment epithelium may be among the earliest detectable signs of inflammatory eye disease. In intermediate uveitis, electroretinography is a valuable complementary test for detection of retinal damage,^{12,13} and the pattern ERG (PERG) is a useful indicator of macular abnormalities.¹⁴ We performed electrophysiological testing in a group of FHC patients, in order to further characterise the extent of ocular involvement.

METHODS

Twenty-one patients (9 males and 12 females; age 15-68 years, mean 37.86 years) with unilateral FHC attending the Uveitis Clinics at the Birmingham and Midland Eye Hospital were examined. Eighteen patients were Caucasian and three of Indo-Pakistani origin. The median duration of disease from diagnosis was 35 months (range 11-137 months). All patients had clinical findings consistent with diagnostic criteria proposed by La Hey *et al.*¹⁵ Only patients with visual acuity ranging from 6/5 to 6/9 in the affected eyes were chosen (Table I). Greater than +1 vitreous opacities in the visual axis was an exclusion criterion.¹⁶ Patients with secondary glaucoma were also excluded from the study, to avoid spurious ERG findings.¹⁷

Of 21 FHC eyes, 10 (48%) were pseudophakic. These patients had undergone extracapsular cataract extraction with heparin surface modified posterior chamber lens implant at least 3 months previously (range 3-74 months; median 22.5 months). They were no longer on any topical treatment and had minimal inflammatory activity at the time of examination. No patient had clinical evidence of post-operative cystoid macula oedema (CMO) or sustained rises in intraocular pressure. All the unaffected eyes were phakic.

Table I. Individual patient data from the 21 patients studied

Patient no.	Visual acuity			Posterior capsule	Scotopic ERG				30 Hz ERG		PERG	
	FHC eyes	Unaffected eyes	Lens		a-wave		b-wave		Amplitude	Latency	Amplitude	Latency
					Amplitude	Latency	Amplitude	Latency				
1	6/5	6/4	Clear	-	-	+	-	+	-	+	-	
2	6/9	6/6	Clear	-	-	+	-	-	-	++	+	
3	6/5	6/6	Clear	+	-	+	-	+	-	-	+	
4	6/6	6/6	Clear	-	-	+	-	-	-	++	+	
5	6/6	6/6	Clear	-	-	-	-	+	+	-	+	
6	6/9	6/9	Clear	-	-	-	-	+	+	++	+	
7	6/6	6/5	Clear	+	-	+	-	+	-	-	+	
8	6/5	6/5	PSCLO	+	-	+	+	+	+	++	+	
9	6/9	6/5	PSCLO	-	-	-	-	-	-	++	+	
10	6/6	6/4	PSCLO	-	-	-	-	-	-	++	-	
11	6/6	6/6	PSCLO	-	-	-	-	-	-	++	+	
12	6/6	6/4	IOL	Clear	-	-	-	+	+	-	-	
13	6/6	6/6	IOL	Clear	-	-	-	-	-	++	+	
14	6/9	6/5	IOL	Clear	-	-	-	+	+	++	+	
15	6/9	6/6	IOL	Clear	++	-	++	-	++	-	++	
16	6/9	6/4	IOL	Opacity	-	-	-	-	-	-	-	
17	6/9	6/6	IOL	Opacity	-	-	+	+	+	+	+	
18	6/9	6/4	IOL	Opacity	+	-	+	-	+	-	+	
19	6/5	6/9	IOL	Opacity	-	-	-	-	-	+	-	
20	6/6	6/5	IOL	Opacity	-	-	-	-	+	+	-	
21	6/9	6/4	IOL	Opacity	-	-	-	-	+	++	+	

PSCLO, posterior subcapsular lens opacity; IOL, intraocular lens. Amplitude/latency: -, normal; +, abnormal ($\geq 120\%$ $\leq 150\%$) of normal fellow eye; ++, grossly abnormal ($>150\%$ of normal fellow eye).

All subjects were refracted to their best corrected Snellen visual acuities. Pupillary responses were tested. Slit lamp examination of the anterior segment was performed and intraocular pressures measured. Pattern electroretinography was undertaken followed by pupillary dilation with tropicamide 1%. Flash ERGs were then recorded, following which a detailed retinal examination was carried out.

Electrophysiological Testing

PERGs were performed using a checkerboard stimulus of spatial frequency 0.5 cycles per degree (cpd) and 1.0 cpd, reversing at 6 Hz (100% contrast). Appropriate corrective lenses were used during testing. All pseudophakes and patients with lens opacity were excluded when analysing the PERG results.

Flash electroretinography was performed using a standardised Ganzfeld stimulus. The patients were dark-adapted for 35 minutes. Full-field Ganzfeld dome stimulation was used and responses assessed using carbon fibre electrodes in contact with the cornea. Reference electrodes were placed at the outer canthi. As some of the patients had media opacities the intensities used were higher than those recommended by the International Standardisation

Committee.¹⁸ Our standard flash under scotopic conditions was therefore 2.8 foot-lamberts. To assess the effects of media opacity, an intense flash of 28 foot-lamberts was also employed. As the a-wave latency and amplitude are still changing at these stimulus intensities (the b-wave having saturated), the a-waves between the normal and FHC eyes were compared at standard and intense flash.

Oscillatory potentials were measured by digitally filtering the standard flash response between 100 and 1000 Hz. Cone and rod oscillatory potentials were identified and analysed.¹⁸ Photopic and 30 Hz ERGs were recorded using stimulus intensities of 20 and 5 foot-lamberts respectively, against a background of 20 foot-lamberts.

Our laboratory's reference values show a maximum interocular variation of 10% for flash ERG and 20% for PERG (p50 and n95 components). For b-wave latency, the normal interocular limit is ± 0.8 ms. Other reference values, based on recordings from 100 normal individuals aged between 15 and 60 years, are shown in Table II. None of these normals was pseudophakic.

Table II. Laboratory reference values for flash ERG and PERG

	Scotopic ERG			PERG (mean \pm 2.5 SD)	
	a-wave	b-wave	30 Hz ERG	p50	n95
Amplitude (μ V)	-	-	25-120	2.60 \pm 1.25	3.50 \pm 1.35
Standard flash (SF)	50-145	290-800	-	-	-
1 log>SF	110-240	290-800	-	-	-
Latency (ms)	-	-	26-33	-	-
Standard flash (SF)	20.5-24.8	-	-	-	-
1 log>SF	11.5-14.5	-	-	-	-

Table III. Comparison between the number of abnormal findings in phakic eyes with FHC and pseudophakic eyes with FHC

Result	Scotopic ERG		30 Hz ERG	PERG
	a-wave No. (%)	b-wave No. (%)		
Normal				
Phakic (<i>n</i> = 11)	8 (73)	5 (45)	5 (45)	1 (9)
Pseudophakic (<i>n</i> = 10)	8 (80)	7 (70)	3 (30)	2 (20)
Significance ^a	<i>p</i> = 1.0	<i>p</i> = 0.39	<i>p</i> = 0.66	<i>p</i> = 0.59
Reduced				
Phakic (<i>n</i> = 11)	3 (27)	6 (54)	6 (54)	7 (64)
Pseudophakic (<i>n</i> = 10)	2 (20)	3 (30)	5 (50)	7 (70)
Significance ^a	<i>p</i> = 1.0	<i>p</i> = 0.39	<i>p</i> = 1.0	<i>p</i> = 1.0
Delayed				
Phakic (<i>n</i> = 11)	0 (0)	1 (9)	3 (27)	9 (82)
Pseudophakic (<i>n</i> = 10)	0 (0)	1 (10)	5 (50)	6 (60)
Significance ^a	—	<i>p</i> = 1.0	<i>p</i> = 0.39	<i>p</i> = 0.36

^aFisher's exact test.

Statistical analysis was by the paired Student's *t*-test for continuous data and by the Fisher's exact test for categorical data.

RESULTS

Of the 21 eyes with FHC, 11 (52%) were phakic and 10 (48%) pseudophakic (Table I). Dilated funduscopy was normal in all 21 patients. Table III shows a comparison of electroretinographic findings between the phakic and pseudophakic eyes with FHC. There was no significant difference between these two groups.

The ERG scotopic a-wave was abnormal in only 5 patients, of whom 3 (60%) had no lens or posterior capsule opacity. The scotopic b-wave was abnormal in 9 patients, 6 (67%) of whom had no lens or posterior capsule opacity. In addition, 30 Hz ERG abnormalities occurred in 6 phakic patients, of whom 5 (83%) had no lens opacity (Tables I, III, IV). Lens opacities, if sufficiently dense, cause a neutral-density filter effect with reduction and delay of the a- and b-waves of the flash ERG.¹⁹ As these results suggest that media opacities did not significantly affect the flash ERG results, all flash ERG results were analysed.

Flash ERG

There was no significant difference in the mean a-wave amplitude or latency between the normal eyes and the eyes with FHC at either stimulus intensity (Table V). Scotopic b-wave amplitudes were reduced in 9 (43%) of 21 FHC eyes (Fig. 1). The mean b-wave amplitude of the FHC eyes was significantly reduced compared with the normal eyes. This reduction was to both standard and intense flash stimulation (*p* = 0.036 and *p* = 0.021, respectively). Delay of the scotopic b-wave was noted in 2 (10%) of 21 eyes. There was no significant difference in mean latency of the scotopic b-wave between the FHC eyes and the control group. The 30 Hz ERGs were reduced in 11 (52%) of the 21 FHC patients (Fig. 2), and delayed in 8 (38%) of 21 FHC eyes. The mean 30 Hz ERG amplitude of the 21 FHC eyes was significantly reduced as compared with normal fellow eyes (*p* = 0.006). There was no significant difference in mean latency. Oscillatory potentials were reduced in 15 (71%) of 21 FHC eyes (Figs. 1, 2). The most marked reductions involved those arising from the cone system. The mean amplitude of the oscillatory potentials was significantly reduced in the FHC eyes compared with the fellow eyes (*p* = 0.006 for rod system, *p* = 0.001 for cone system). All eyes with a

Table IV. Comparison of the number of abnormal findings in phakic eyes with and without lens opacity

Result	Scotopic ERG		30 Hz ERG	PERG
	a-wave No. (%)	b-wave No. (%)		
Normal				
Clear lens (<i>n</i> = 7)	5 (71)	2 (29)	2 (29)	1 (14)
Opacity (<i>n</i> = 4)	3 (75)	3 (75)	3 (75)	0 (0)
Significance ^a	<i>p</i> = 1.0	<i>p</i> = 0.24	<i>p</i> = 0.24	<i>p</i> = 1.0
Reduced				
Clear lens (<i>n</i> = 7)	2 (29)	5 (71)	5 (71)	3 (43)
Opacity (<i>n</i> = 4)	1 (25)	1 (25)	1 (25)	4 (100)
Significance ^a	<i>p</i> = 1.0	<i>p</i> = 0.24	<i>p</i> = 0.24	<i>p</i> = 0.19
Delayed				
Clear lens (<i>n</i> = 7)	0 (0)	0 (0)	2 (29)	6 (86)
Opacity (<i>n</i> = 4)	0 (0)	1 (25)	1 (25)	3 (75)
Significance ^a	—	<i>p</i> = 0.36	<i>p</i> = 1.0	<i>p</i> = 1.0

^aFisher's exact test.

Table V. Comparison between the mean flash ERG amplitude and latency in 21 patients

Component	FHC eyes (SD)	Unaffected eyes (SD)	Significance ^a
a-wave SF scotopic Amplitude (μV)	81.50 (30.61)	94.71 (35.65)	$p = 0.090$
Latency (ms)	23.41 (14.39)	22.80 (14.53)	$p = 0.341$
a-wave 1 log>SF Amplitude (μV)	148.81 (47.66)	174.41 (71.49)	$p = 0.069$
Latency (ms)	14.13 (7.42)	13.74 (7.15)	$p = 0.221$
b-wave SF scotopic Amplitude (μV)	154.60 (22.50)	189.30 (30.20)	$p = 0.036$
Latency (ms)	53.70 (15.72)	51.30 (18.42)	$p = 0.177$
b-wave 1 log>SF Amplitude (μV)	219.06 (13.43)	262.75 (17.83)	$p = 0.021$
Latency (ms)	46.50 (13.01)	44.80 (11.87)	$p = 0.092$
30 Hz ERG Amplitude (μV)	39.60 (16.12)	52.40 (19.20)	$p = 0.006$
Latency (ms)	29.40 (10.86)	27.80 (10.40)	$p = 0.193$
Oscillatory rod	15.10 (16.08)	21.60 (16.63)	$p = 0.006$
Oscillatory cone	9.03 (7.56)	16.40 (11.14)	$p = 0.001$

SF, standard flash.

^aPaired Student's *t*-test.

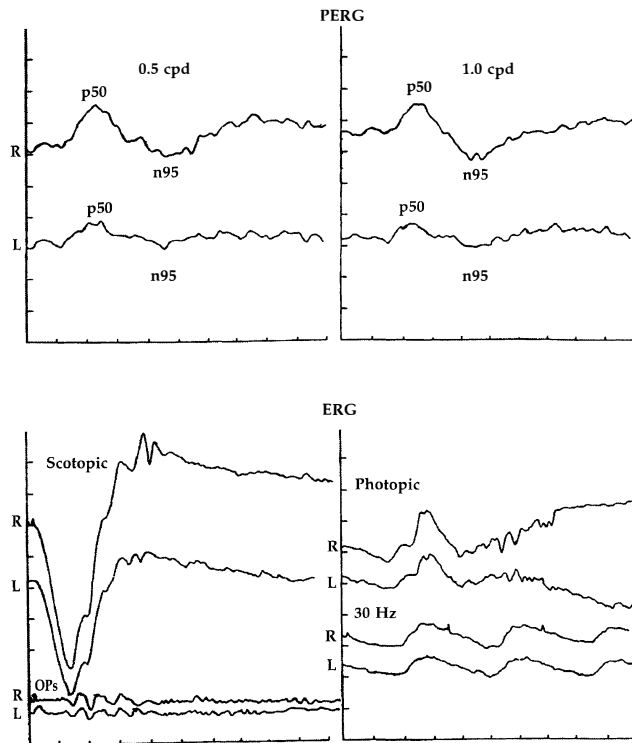


Fig. 1. A 22-year-old woman with left FHC (patient no. 4, Table I). Both eyes were phakic with clear media. Right VA 6/6; left VA 6/6. There is selective reduction of the scotopic b-wave with reduced PERG in the left eye. Sensitivity and sweep times per division: PERG, 2.49, μV and 20.0 ms; ERG, 49.79, μV and 10.0 ms.

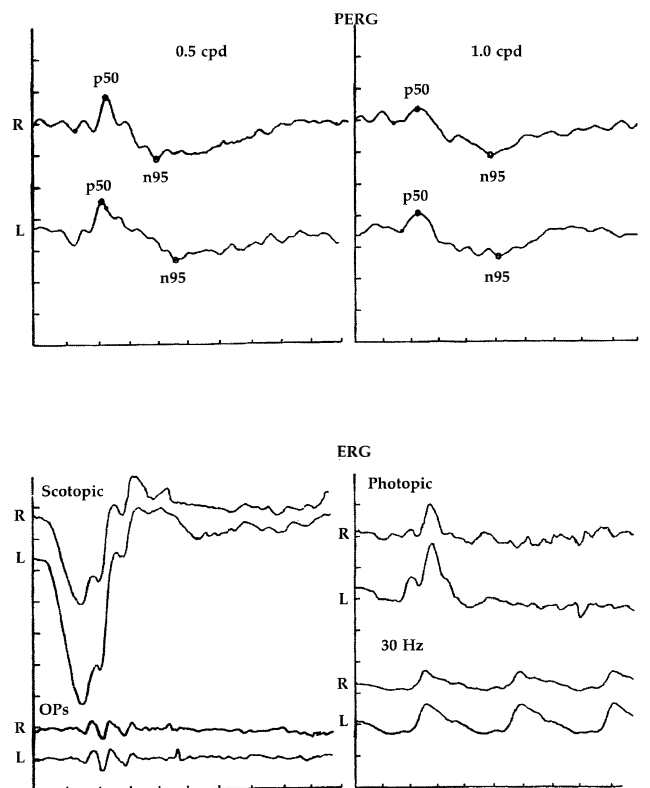


Fig. 2. A 33-year-old man with right FHC (patient no. 3, Table I). Both eyes were phakic with clear media. Right VA 6/5; left VA 6/6. There is a reduced ERG in the right to scotopic, photopic and 30 Hz stimulation. Slightly reduced oscillatory potentials (OPs) and a slightly reduced and delayed PERG in the right eye are seen. Sensitivity and sweep times per division were as in Fig. 1.

Table VI. Comparison between mean PERG amplitude and latency in 7 phakic patients without lens opacity with FHC

Component	FHC eyes (SD)	Unaffected eyes (SD)	Significance ^a
0.5 cpd			
Amplitude (μV)			
p50	2.01 (0.24)	2.44 (0.69)	$p = 0.047$
n95	3.46 (0.63)	3.92 (0.53)	$p = 0.074$
Latency (ms)			
p50	50.40 (4.29)	43.80 (3.97)	$p = 0.005$
n95	93.80 (5.37)	85.90 (5.93)	$p = 0.012$
1.0 cpd			
Amplitude (μV)			
p50	1.73 (0.34)	2.64 (0.56)	$p = 0.003$
n95	2.86 (0.58)	3.56 (0.66)	$p = 0.027$
Latency (ms)			
p50	51.6 (4.13)	44.2 (4.71)	$p = 0.004$
n95	94.6 (7.38)	86.9 (6.72)	$p = 0.032$

cpd, cycle per degree.

^aPaired Student's *t*-test.

reduction in oscillatory potentials also had a reduction of PERG to both 0.5 and 1.0 cpd check stimuli.

Pattern ERG

A total of 18 FHC eyes (86%) exhibited abnormal PERGs. The uninvolved eyes all showed normal responses. All 4 phakic FHC eyes with lens opacity had grossly reduced ($<1 \mu\text{V}$) PERGs. Of those FHC eyes without lens opacity ($n = 7$), 3 (43%) had grossly reduced PERGs. For these reasons only the 7 phakic patients with clear lenses were included for comparison of the PERG with the normal fellow eye

(Table VI; Figs. 3, 4). Table VI shows that to a 0.5 cpd stimulus there was significant reduction in amplitude of the p50 component ($p = 0.047$) and increased latency of both p50 and n95 components ($p = 0.005$ and $p = 0.012$ respectively) in the FHC eyes. To a 1.0 cpd stimulus both p50 and n95 were significantly reduced in amplitude ($p = 0.003$ and $p = 0.027$, respectively) as well as significantly delayed ($p = 0.004$ and $p = 0.032$, respectively) in the FHC eyes (Figs. 3, 4). Four of 7 (57%) phakic eyes with clear media had reduced PERG to less than 80% of the normal fellow eye. Three of these 4 had

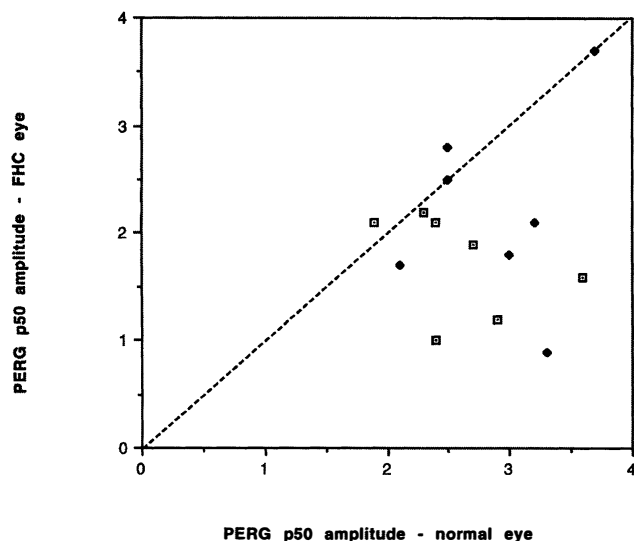


Fig. 3. Scattergram showing individual paired results of PERG p50 amplitude (in mV) in 7 phakic patients with clear media. Filled circles, 0.5 cycle per degree; open squares, 1.0 cycle per degree. Points below the dotted line indicate reduced amplitude in the eye with FHC compared with the normal fellow eye.

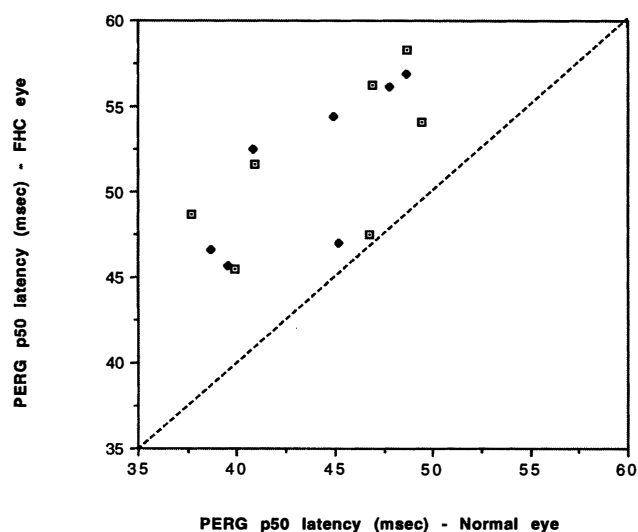


Fig. 4. Scattergram showing individual paired results of PERG p50 latency in 7 phakic patients with clear media. Filled circles, 0.5 cycle per degree; open squares, 1.0 cycle per degree. Points above the dotted line indicate increased latency in the eye with FHC compared with the normal fellow eye.

reduction in amplitude of the PERG to a 1.0 cpd check stimulus to less than 50% that of the normal fellow eye.

Visual Acuity (VA)

The VA in 8 eyes with FHC was as good as or better than that in the fellow eye (Table I). The amplitude of the PERG was reduced in 6 of these 8 eyes (75%), 4 of these 6 (67%) having p50 components of $<1 \mu\text{V}$ amplitude. Flash ERG abnormalities occurred in 5 of the 8 eyes (62%). In those FHC eyes with reduced VA as compared with the fellow eye, 8 of 13 eyes (62%) with FHC had reduction of the PERG. Five of these 8 eyes (62%) had amplitude reduction to $<1 \mu\text{V}$. Flash ERG abnormalities occurred in 10 of the 13 eyes (77%). There was no relationship between reduced VA and ERG abnormalities.

DISCUSSION

Our results show evidence of damage to the inner retinal layers (inner plexiform layer) in FHC eyes with good visual acuities. Martenet and Niemeyer¹³ also found a reduction of the flash ERG potentials in eyes with intermediate uveitis and good vision. In FHC they found the flash ERG normal or only slightly altered. There is a normal interocular variation in amplitude of the signal of the ERG (mean variation from a normal sample between fellow eyes) but this is usually small. Allowing for this, the reduction in amplitude and delay in the latency of the PERG and the reduction in amplitude of the flash ERG in our study were statistically significant.

A major factor that may have affected the ERG results is the presence of media opacity. All patients chosen for the study had good vision (6/5–6/9). Because the uninvolved eyes served as the control group this was considered essential, as it is well recognised that the signal of the photopic ERG is affected by relative brightness of stimulus. Bright, direct light produces faster, higher-amplitude ERGs than those produced by less bright stray light. Accurate focusing of the pattern is also essential in order to obtain an accurate PERG. Lens opacities may have reduced the amplitude of the PERG due to difficulties in focusing the pattern. For these reasons patients were selected to minimise the effects of opacities of the media on the flash ERG and PERG and all pseudophakic patients and patients with lens opacity were excluded when analysing the PERG results. Significant vitreous opacities in the visual axis was also an exclusion criterion and no fundal abnormalities could be detected in any of the eyes studied.

Lens opacities, if sufficiently dense, cause a neutral-density filter effect with reduction and delay of the a- and b-waves of the flash ERG. At the range

of intensities used in this study, the a-wave should be more affected by 1 log unit changes in intensity than the b-wave, which saturates at lower intensities than the a-wave.²⁰ Any reduction or delay of the a-wave in our results would therefore indicate a significant neutral-density effect, especially if there was a change with change in stimulus intensity. There was, however, no significant difference in the mean a-wave amplitude or latency between normal eyes and the eyes with FHC at either standard flash intensity (SF) or $1 \log > \text{SF}$. In addition, there was no significant difference in the incidence of flash ERG abnormalities between FHC eyes with VA as good as or better than that in the fellow eye and those with reduced VA as compared with the fellow eye. Opaque media was therefore not considered a significant factor affecting the flash ERG.

Before the results could be attributed to retinal or macular pathology associated with FHC it was necessary to rule out other causes for these changes. Traumatic damage to the eye affects all electrodiagnostic tests, but the a- and b-waves of the ERG should only be affected by severe injuries.²¹ Ten (48%) of 21 eyes in our study were pseudophakic. No patient had undergone Nd:YAG laser capsulotomy. High intraoperative retinal light exposure has been reported as causing substantial reduction of electrophysiological potentials,²² but light protection using the filter on modern operating microscopes and the short operating time required to perform cataract surgery can prevent deterioration of ERG potentials. Cataract extraction may result in post-operative CMO, yet the incidence of CMO in patients with FHC undergoing cataract surgery is negligible, in sharp contrast to the general situation for uveitis patients.^{23–25} Clinically significant CMO has been defined as a drop in vision from a prior visit of one or more Snellen lines associated with ophthalmoscopically visible cystoid changes in the macula or evidence of cystoid changes on fluorescein angiography.²⁶ Although none of our pseudophakic patients had clinically significant CMO, subclinical changes can only be completely excluded by fluorescein angiography. The routine use of this potentially hazardous procedure in these eyes, however, could not be clinically justified. Salzman *et al.*²⁷ showed no significant difference in electrophysiological data between aphakic eyes and those with intraocular lenses, but no comparison between phakic and aphakic or between phakic and pseudophakic patients was made. In our study, comparison between phakic and pseudophakic patients showed no significant difference in the ERG.

La Hey *et al.*²⁸ reported a high incidence of a positive cellular autoimmune response to retinal S-antigen in patients with FHC compared with healthy controls and patients with other types of anterior

uveitis. Our electroretinographic findings suggest that patients with FHC may have subclinical retinal damage. Autoimmunity directed against retinal or choroidal antigens has been suggested to play a role in the chorioretinal lesions observed in patients with FHC.^{8,28} It is possible that prior exposure of eyes with FHC to retinal S-antigen results in sensitisation, and induces an autoimmune breakdown of the blood-retinal barrier. Light microscopy has disclosed abnormal hyalinisation and occasionally endothelial proliferation of iris vessel walls, with narrowing of the vessel lumen.²⁸ This may ultimately lead to occlusion, which in turn may result in subclinical damage.

The PERG abnormalities, particularly the reduction and delay of p50, indicate preganglionic retinal abnormalities within the macular region in eyes with FHC. Furthermore, the selective reduction of the scotopic b-waves, and the reduced oscillatory potentials, indicate some abnormality involving the inner retinal layers, including bipolar cells and amacrine cells, especially those involving the cone system. This is further substantiated by the abnormal 30 Hz ERGs. Although 10 of 21 patients (48%) had some lens or posterior capsule opacity, the normal a-wave rules out any changes in the flash ERG due to opaque media.²⁰

Our findings suggest subclinical retinal damage in eyes with FHC, principally involving the macula. There is also evidence of more widespread inflammatory retinal change involving the bipolar and Müller cell layers. The photoreceptors appear to be largely unaffected since the a-wave of the scotopic ERG was usually normal.

Key words: Fuchs' heterochromic cyclitis, Electrophysiology, Electroretinogram, b-wave, Retina.

REFERENCES

- Jones NP. Fuchs' heterochromic uveitis: an update. *Surv Ophthalmol* 1993;37:253-72.
- La Hey E, de Jong PTVM, Kijlstra A. Fuchs' heterochromic cyclitis: review of the literature on the pathogenetic mechanisms. *Br J Ophthalmol* 1994;78:307-12.
- Murray PI, Hoekzema R, Luyendijk L, Konings S, Kijlstra A. Analysis of aqueous humor immunoglobulin G in uveitis by enzyme linked immunosorbent assay, isoelectric focusing and immunoblotting. *Invest Ophthalmol Vis Sci* 1990;3:2129-35.
- Murray PI, Hoekzema R, van Haren MAC, Luyendijk L, Kijlstra A. Aqueous humour analysis in Fuchs' heterochromic cyclitis. *Curr Eye Res* 1990;9(Suppl):53-7.
- Murray PI, Young DW. Soluble interleukin-2 receptors in retinal vasculitis. *Curr Eye Res* 1992;11(Suppl):193-5.
- de Abreu M, Belfort R, Hirata P. Fuchs' heterochromic cyclitis and ocular toxoplasmosis. *Am J Ophthalmol* 1982;93:739-44.
- Saroux H, Laroche L, Le Hoang P. Secondary Fuchs' heterochromic cyclitis: a new approach to an old disease. *Ophthalmologica* 1985;190:193-8.
- Arffa RC, Schlagel TF. Chorioretinal scars in Fuchs' heterochromic iridocyclitis. *Arch Ophthalmol* 1984;102:1153-5.
- La Hey E, Rothova A. Fuchs' heterochromic cyclitis in congenital ocular toxoplasmosis. *Br J Ophthalmol* 1975;75:372-3.
- La Hey E, Baarsma GS. Contralateral active ocular toxoplasmosis in Fuchs' heterochromic cyclitis. *Br J Ophthalmol* 1993;77:455-6.
- Jones NP. Fuchs' heterochromic uveitis: a reappraisal of the clinical spectrum. *Eye* 1991;5:649-61.
- Martenet AC. Intermediate uveitis. *Bull Soc Belge Ophthalmol* 1989;230:33-9.
- Martenet AC, Niemeyer G. The value of electroretinography in uveitis. *Ophthalmologie* 1990;4:169-72.
- Arden GB, Carter RM, Macfarlan A. Pattern and Ganzfeld electroretinograms in macular disease. *Br J Ophthalmol* 1984;68:878-84.
- La Hey E, Baarsma GS, De Vries J, Kijlstra A. Clinical analysis of Fuchs' heterochromic cyclitis. *Doc Ophthalmol* 1991;78:225-35.
- Nussenblatt RB, Palestine AG, Chan C-C, Roberge F. Standardization of vitreal inflammatory activity in intermediate and posterior uveitis. *Ophthalmology* 1985;92:467-71.
- Weinstein G, Arden GW, Hitchings RA, Ryan S, Calthorpe CM, Odom JV. The pattern electroretinogram (PERG) in ocular hypertension and glaucoma. *Arch Ophthalmol* 1988;106:923-8.
- International Standardization Committee. Standard for clinical electroretinography. *Arch Ophthalmol* 1989;107:816-9.
- Babel J, Stangos N, Korol S, Spiritus M. Ocular electrophysiology. Stuttgart: Georg Thieme, 1977, 11-3.
- Ikeda H. Clinical electroretinography: The ERG as a function of flash intensity. In: Halliday AM, editor. Evoked potentials in clinical testing. Edinburgh: Churchill Livingstone, 1993:126-9.
- Crews SJ, Thompson CRS, Harding GFA. The ERG and VEP in patients with severe eye injury. *Doc Ophthalmol Proc Ser* 1978;15:203-9.
- Lessel M, Thaler A, Heilig P, Jantsch W, Scheiber V. Intraoperative retinal light damage reflected in electrophysiological data. *Doc Ophthalmol* 1991;76:323-33.
- Foster CS, Fong LP, Singh G. Cataract surgery and intraocular lens implantation in patients with uveitis. *Ophthalmology* 1989;96:281-7.
- Hooper PL, Rao NA, Smith RE. Cataract extraction in uveitis patients. *Surv Ophthalmol* 1990;35:120-44.
- Stavrou P, Murray PI. Heparin surface modified intraocular lenses in uveitis. *Ocular Immunol Inflamm* 1993;4:309-14.
- Stark WJ Jr, Maumenee AE, Fagadau W, Datiles M, et al. Cystoid macular edema in pseudophakia. *Surv Ophthalmol* 1984;28 (Suppl): 442-51.
- Salzman J, Seiple W, Carr R, Yannuzzi L. Electrophysiological assessment of aphakic cystoid macular oedema. *Br J Ophthalmol* 1986;70:819-24.
- La Hey E, Broersma L, van der Gaag R, Baarsma GS, Rothova A, Kijlstra A. Does autoimmunity to S-antigen play a role in Fuchs' heterochromic cyclitis? *Br J Ophthalmol* 1993;77:436-9.
- Goldberg MF, Erozen YS, Duke JR, Frost JK. Cytopathologic and histopathologic aspects of Fuchs' heterochromic cyclitis. *Arch Ophthalmol* 1965;74:604-9.